

Taylor (R.W.) & Van Gieson (I.)

OBSERVATIONS ON PRURIGO

CLINICAL AND PATHOLOGICAL

BY

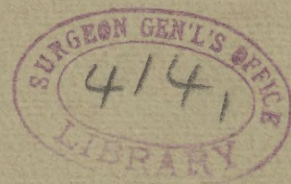
R. W. TAYLOR, M. D.

CLINICAL LECTURER ON VENEREAL DISEASES AT THE COLLEGE OF PHYSICIANS AND SURGEONS, NEW YORK

AND

IRA VAN GIESON, M. D.

FIRST ASSISTANT AT THE LABORATORY OF THE ALUMNI ASSOCIATION OF THE
COLLEGE OF PHYSICIANS AND SURGEONS



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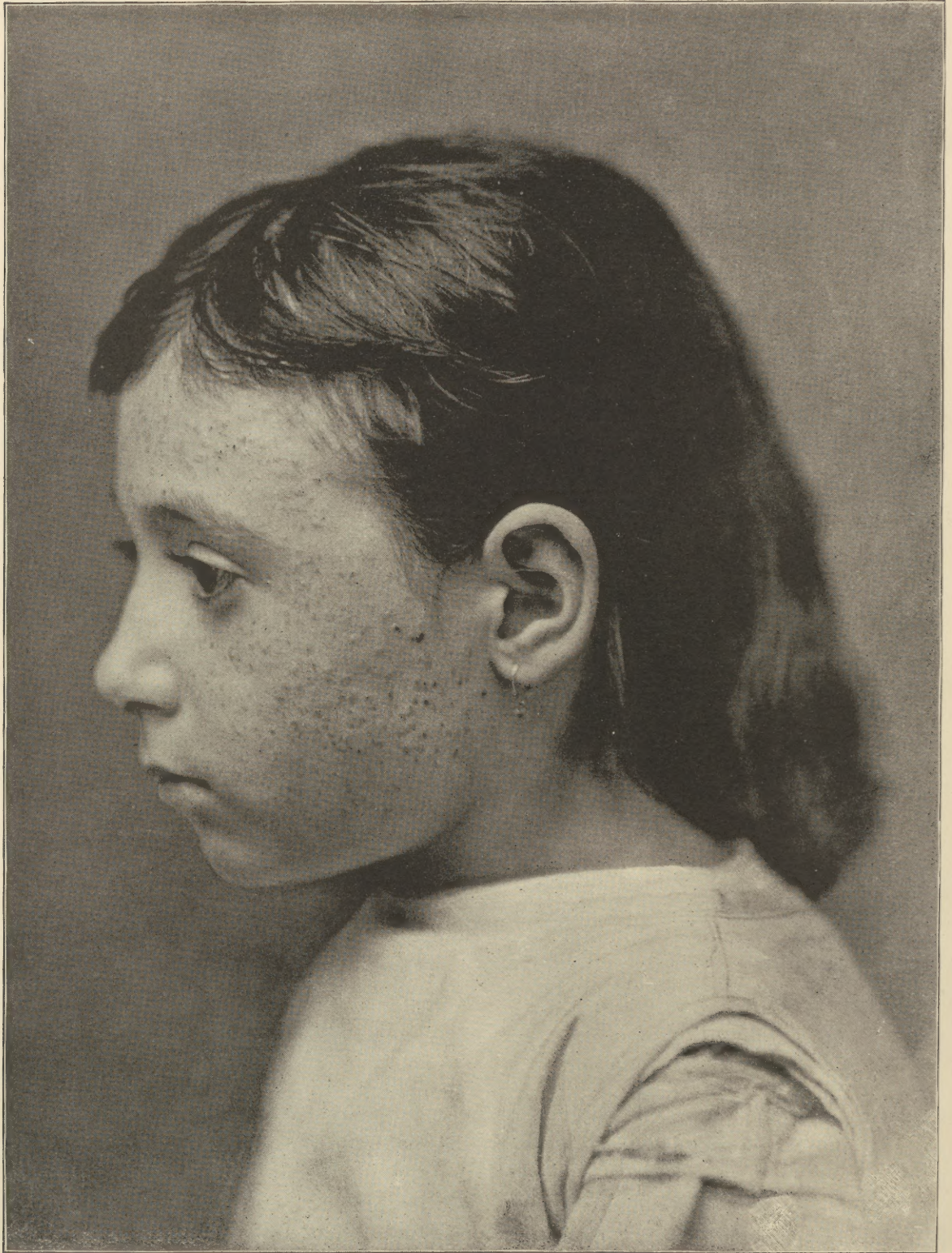


FIG. 1.—Showing prurigo papules on the forehead and cheek.

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OBSERVATIONS ON PRURIGO, CLINICAL AND PATHOLOGICAL.*

THE great interest shown in the discussion of Dr. Zeisler's paper on prurigo at our meeting last year has prompted me to prepare this essay, which will deal with the clinical aspects and pathological changes offered by that disease. Those of the older members now present will remember that at our first meeting, on the occasion of the reading of the history of a case of prurigo by Dr. R. Campbell, in the recital of their experience, the combined members could only produce six cases. Thirteen years then elapsed before the disease was again brought before this association, and then by a new member, who, thoroughly skilled in the recognition of the disease, detailed twelve cases which he had observed in Chicago in private and public practice during a period of five years. His experience certainly warranted his statements, first, that prurigo did surely exist in America; second, that the severe form prurigo ferox seu agria, while occurring in perfectly typical examples and on patients born and reared here, was of comparative rarity and was often only imported; and, third, that prurigo mitis was not uncommon here. In the discussion of this paper the members reiterated their opinion of the rarity of the disease, and, all told, gave eighteen cases as the aggregate of their experience of well-remembered cases, though the existence of a few more was hinted at. Seeing that six of the participants in the discussion last year had given their combined experience of six cases thirteen years previously, the number of new cases then cited was certainly very small when it is remembered that there were ten gentlemen present who were not members and had not been present at the first meeting. These facts will certainly prove that prurigo is, taking the aggregate of cases of skin diseases, rather rare in America. Is it, however, as rare as it was commonly supposed? Dr. Zeisler's showing is certainly contrary to such a view, and I think myself that many cases escape recognition and are classed as eczema, scabies, pediculosis, ecthyma, impetigo, and even ichthyosis. Dr. Bronson very aptly remarked last year that a bug-bear had been made of this disease, and I think he is right. I have many times witnessed the diffidence and want of confidence in their diagnosis of prurigo by physicians speaking of a personal case, and the skepticism and even incredulity of their hearer or hearers. This deep-rooted opinion that prurigo is so rare as almost to be unknown in America has, I have no doubt, been the cause of many cases being overlooked and wrongly diagnosticated. Then, again, the constant changes, modifications, and complications which are so common in the course of the disease have, no doubt, very often rendered its diagnosis difficult or even impossible.

But there is another very cogent reason why the disease escapes recognition or is wrongly diagnosticated—namely, that the profession at large has not been educated to a clear

idea of what it is, and that there are no plates or drawings accessible in this country which will aid them in obtaining a good idea of its clinical features. In the descriptions of this disease in our text-books (and they are drawn from or based upon Hebra's writings) much stress (and that rightly) is laid upon the intercurrent dermal affections which complicate prurigo, and on its resemblance at times to scabies, pediculosis, chronic eczema, ichthyosis, etc. The result of all this elaboration—necessary, it is true—is to bewilder a man not well versed in dermatology. What has been needed, in my judgment, in this country, where from the rarity of prurigo our clinical teachers have been unable to present typical cases to students, is a clearly marked drawing of the disease in its uncomplicated state, which shall serve as a basis of study and of elaboration. This I am now fortunate enough to be able to present. In this paper I shall detail the clinical features of quite a severe case of prurigo which is under treatment at my clinic at the New York Hospital, and show three photographs presenting graphic representations of the typical features of the disease in an uncomplicated condition. A study of the history of the case and of the drawings, I am sure, will be of benefit to any one desiring to familiarize himself with the clinical picture of prurigo proper. I shall also allude to the usual concomitants and modifying conditions during the course of the disease, and try to make clear how to recognize it even when marked by intercurrent morbid conditions.

The patient is a girl, about nine years old, thin and weakly, the offspring of healthy American parents who are in good circumstances and accustomed to good food and healthful surroundings. She is the oldest of three children. She was well, but not robust, in her earlier years, and when four years of age it was noticed that she began to scratch. At this time she was spending the summer on Staten Island, and the appearance of little red pimples and larger patches on the face, forearms, and legs led the parents to think that she was the victim of mosquitoes, as these insects were then numerous and rapacious. Close questioning of the mother led me to the conclusion that the initial eruption was urticaria or lichen urticatus. Beginning in hot weather, the eruption of wheals and the pruritus went hand in hand through the fall and winter, and ended at the onset of hot weather in the following summer. The health of the child just prior to the onset of the disease was very good indeed, and her photograph taken then shows her to have been fat and chubby. She was carefully nurtured and regularly bathed. During the first summer and fall of her sickness she seemed well, notwithstanding that her sleep was disturbed at night by itching. In this way the disease had gone on ever since, being, as compared with other and graver cases, moderately severe in winter and ceasing almost entirely during warm weather. During the existence of the disease the child's suffering from pruritus was great, but was subject to periods of exacerbation and of moderation.

The following condition was observed when she came to my clinic at the New York Hospital early in January of this year. The expression of the child's face was rather dull and was typical, in its white, waxen, somewhat ashy hue, of prurigo. Over the forehead, temporal regions, and cheeks was a copious erup-

* Read before the American Dermatological Association at its fourteenth annual meeting, September 2, 1890.

tion of small, conical papules, some whiter than the skin itself, others of a rather yellowish hue, and a few others again capped with a minute blood crust, the result of scratching. The appearance of the eruption was highly suggestive of comedones, but the central black plug of the latter lesions was absent and the blood crusts showed that the eruption caused scratching. Minute examination of the papules showed clearly that they were not developed on the site of sebaceous glands. In Fig. 1 the appearance of the eruption upon the face is admirably well shown. There was no marked dryness and want of vitality in the hair as shown by its dullness and roughness, as I have seen in severer cases, nor was there evidence of so much pityriasis capitis in the slight, mealy desquamation as we sometimes see. As pointed out by Hebra, the neck and nucha were not involved in the disease, but it began to develop where the shoulders merge into the neck. The eruption extends from the bases of the fingers on the backs of the hands to the elbow. It has been localized for a number of years upon the extensor surface of the forearms, entirely sparing the bend of the elbow, but at this time it showed a tendency to spread sparsely upon the flexor surface. The arms, as is the case in the mild form of prurigo, are not so much affected as the forearms, and only on their extensor and outer surfaces. A very typical picture of the disease is shown in Fig. 2 and in the very accurate waxen cast of the arm kindly made for me by Dr. F. J. Levisur. The papules are abundant on the backs of the hands and forearms, are discretely isolated and irregularly scattered, without order or semblance of grouping. They are conical in shape, and as firm in structure as the skin itself. Some of them are of the color of the skin, others have a little reddish hue, while others, from scratching, are capped with a minute blood crust. The skin of the hands and forearms is rather darker than normal, and the natural furrows are a little deeper and wider than normal. A mild, mealy desquamation is sometimes seen by the mother. The appearance of the papules in Fig. 2 is rendered more distinct and salient by the use of a hand glass. I can imagine no better artificial picture of mild prurigo than is shown in Figs. 1 and 2. They certainly afford a groundwork for study of this curious disease in its less advanced, I may almost say earlier, phases, though this child has suffered for several years with it.

At the early examinations of the case it was noted that the disease had skipped from the shoulders to the buttocks, where the papules are rather larger and more sparsely distributed, and the scratch marks and blood crusts on these regions tell the story of vigorous scratching. Over the outer and extensor surfaces of the thighs there are few papules, and the popliteal spaces present a normal appearance. The legs were especially worthy of study. On their outer and anterior surface were very many papules irregularly scattered in a discrete manner, and the evidences of scratching were as marked as in cases of pediculosis corporis. The discoloration of the skin and the increased development of its furrows were well marked. The papules here are seen to be quite large, while on the arms they were of the size of hemp-seeds or the head of a large pin, and conical or globose; here on the leg they attained the size of a small split pea, conical, rounded, and flat. Owing to the dependent position of the parts, and undoubtedly to the irritation of scratching, there is considerable interpapular hyperæmia, and toward the ankles evidence of inflammatory œdema, and ecchymatous and blood crusts, large and small, were scattered freely among the papules.

The ganglia in the neck are very much enlarged, those in the axillæ are larger than normal, while those of the epitrochlear and inguinal regions can be distinctly felt to be increased in size. The typical prurigo buboes, of walnut size and even fist

size, were not seen in this case, and could scarcely be expected in a subject so young having a tolerably mild form of the disease. The amount of adenopathy is usually in proportion to the extent, duration, and severity of the disease. There was an entire absence of lanugo upon the backs of the hands and forearms, and the small hairs of the legs were seen to be broken off near the skin level.

Besides the visible papules, firm pressure with the index-finger tip over the skin of the forearms and legs revealed a shot-like sensation, caused by the presence there of little subepidermal papular masses. Once or twice, when no salient papules were to be seen, this maneuver revealed to the touch the hidden lesions.

From careful and repeated observations and studies I am led to think that the development and course of the papules occur in the following manner: These lesions are first noticed as little shotty bodies under the skin or, seemingly, in the rete. In this stage they may undergo involution and disappear, or they may increase in height and become more or less salient, as shown in Figs. 1, 2, and 3. In the state of salience, if not destroyed by friction or bacterial infection, they may remain unchanged indefinitely or they may undergo involution. In this latter event they then slowly or perhaps rapidly subside, the process being accompanied by mild desquamation, until either no trace of them is left on the skin, or minute shining, very slightly depressed spots of mild atrophy remain to show where the papules once were. This atrophic condition, judging from clinical observation, I should think was not permanent, but Dr. Van Gieson's pathological studies seem to give evidence that it may be permanent.

In this case, as in most others of its class, it was impossible to keep the child under such care and restraint as the gravity of its disease demanded; therefore its chronic course was complicated in various ways. Sometimes, when the treatment was carefully followed, the child's condition was improved. Then, as a result of inattention, of the want of baths, and of carelessness in diet, and perhaps from natural causes, an exacerbation would occur. The course of prurigo in this case, as in the others which I have seen, was peculiarly erratic, for when we expected improvement we sometimes found the reverse and when we had lost hope of benefit from treatment we sometimes unexpectedly observed it. Thus it was that the child had its ups and its downs, but the disease kept on apace.

Fig. 3 shows the child's arms during a period of marked exacerbation, and it is a picture worthy of study in itself and in connection with Figs. 1 and 2. All the appearances are much exaggerated beyond those of Fig. 2. Over the backs of the hands the papules are very plentiful, and much more closely packed than is common. Over the forearms the visible papules and blood-crusts are very numerous, and the finger-tip revealed many hidden ones. The pigmentation was much greater than in Fig. 2, which was taken four months before Fig. 3. This increased pigmentation I assured myself was not in any way due to exposure to the sun's rays, or to heat, or any form of extraneous irritation. The mother, a sensible, observant woman, was clear on the subject that the skin of the child's arms, legs, and face became very dark at times, and then, during periods of comparative quiescence of the disease, it would gradually become lighter, so that during warm weather it frequently, to her eye, presented no temporary cyclical or irregularly appearing abnormality. This retrocession of the papules and of the pigmentation is a very interesting feature of this



FIG. 2.—Showing pale prurigo papules, and some capped with minute blood-crusts.



FIG. 3.—Showing the eruption of prurigo during a period of exacerbation.



FIG. 4.—Showing a localized hypertrophy of the rete Malpighii, containing a sweat-duct, over which the stratum corneum is slightly thickened. *d*, clusters of small round and polygonal cells.



FIG. 5.—A portion of the skin with thickened horny layer. *d*, as in Fig. 4.

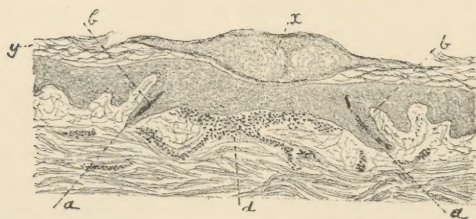


FIG. 6.—Section through a minute pellicle or scab in the horny layer at *aa*. The interpapillary portions of the rete are thrust aside laterally, as if due to pressure from the pellicle. *bb*, sweat-ducts. *dd*, as in Figs. 4 and 5.

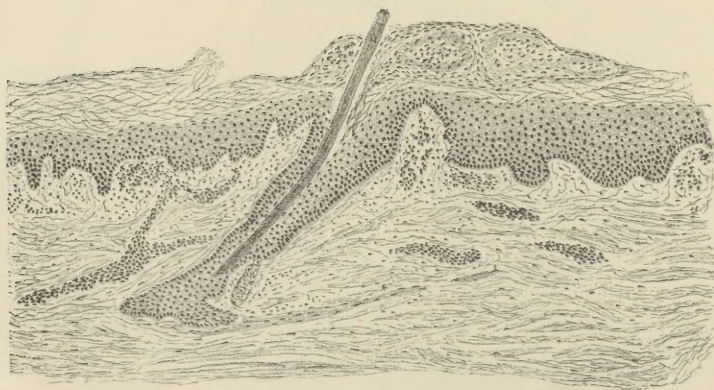


FIG. 7.—Section with a hair passing through the same pellicle or scab shown in Fig. 6. The horny layer is slightly thickened. The hair follicle shows outgrowths of the outer root sheath and atrophy of the sebaceous gland. The cell-clusters, similar to *dd* in Figs. 4, 5, and 6, infiltrate both the papillæ and deeper derma in the section. The arrector pili muscle is shown, and is somewhat hypertrophied.



FIG. 8.—Showing a widening of the intercellular spaces of the lowermost cells, *a*, of the rete Malpighii, as if distended by fluid passing into the rete from the subjacent corium, *b*, whose interfibrillary spaces are distended and œdematous. The rete cells also show a liquefaction of the perinuclear protoplasm, especially in the cell *c*.

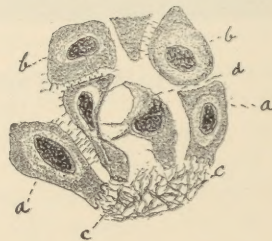


FIG. 9.—Showing a more advanced stage of the distention of the intercellular spaces of the lowermost cells of the rete Malpighii (*a*, lowermost rete cells; *b*, layer of cells just above the lowermost cells; *c*, œdematous subjacent corium). *d*, one of the lowermost rete cells changed into a branching cell surrounded by a large space occasioned by the transudation of fluid from the pars papillaris into the rete Malpighii.

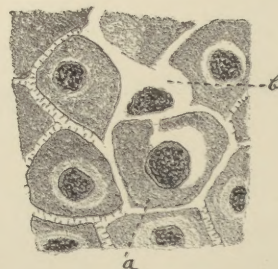


FIG. 10.—From the middle layers of the rete Malpighii, just above the portion represented in Fig. 8. *a*, partially destroyed rete cell. *b*, apparent complete destruction of the cell-body of a rete cell, producing a minute branching cavity communicating with the cavity in the cell *a*. The intercellular spaces of the surrounding cells are somewhat widened, as if distended by fluid.

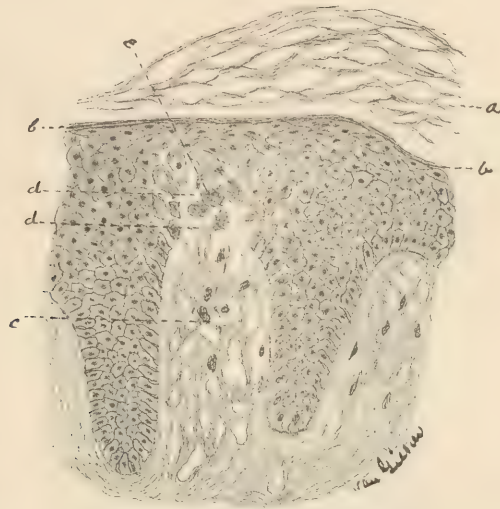


FIG. 11.—A more advanced stage of the degeneration of a group of rete cells and distention of their interspaces, due to the transudation from a corresponding cedematous papilla. *a*, stratum corneum. *b*, stratum lucidum and granulosum. *c*, cedematous papilla. *d*, small branching cavity in the rete Malpighii. *e*, darkly shaded group of rete cells affected with hyaline degeneration. The surrounding pars papillaris is normal.



FIG. 12.—A more highly magnified drawing of the cedematous papilla, and degeneration of the rete cells shown in Fig. 11. *a*, lowermost rete cells; the darkly-colored cells *b*, and portions of the cell *c*, have undergone a hyaline degeneration. *d d d d*, partially degenerated cells.

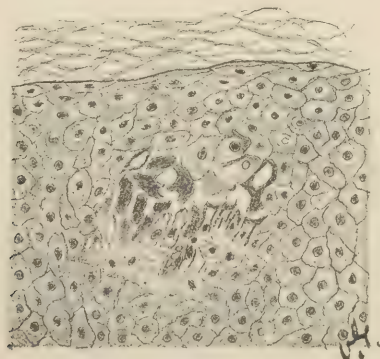


FIG. 13.—Illustrating the hyaline degeneration—indicated by the darkly colored cells—of the rete cells in the middle layers of the epidermis and the formation of a small cyst.

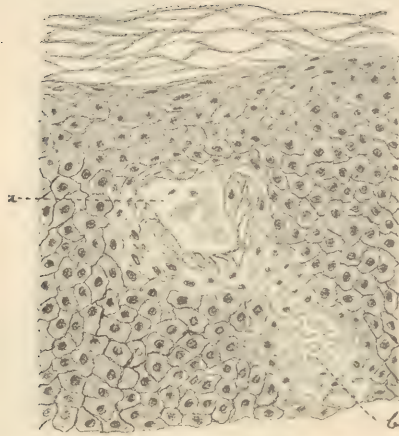


FIG. 14.—Showing the formation of an incipient cavity in the upper layers of the rete Malpighii, by the destruction of the rete cells, due to the transudation of fluid from an underlying œdematous papilla. The cavity at *a* appears to be the result of a more advanced stage of the degeneration of the group of cells at *b*.



FIG. 15.—Showing one of the earlier stages in the formation of the epidermis cysts and the relation of the beginning cysts to the œdematous papillæ. *a*, œdematous papilla. *b*, group of degenerated or liquefying rete cells. *c*, lymph space (or small vein), with swollen endothelial cells. *d*, a small vein surrounded by swollen or proliferating connective-tissue cells and a few cells having the appearance of leucocytes; the situation of this vein is somewhat suggestive as to the source of the fluid exudation distending the interfibrillary spaces of the papilla, *a*.

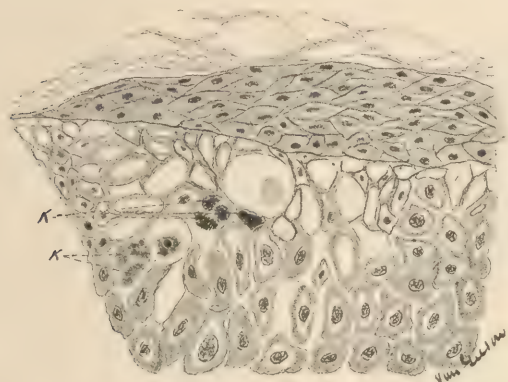


FIG. 16.—A portion of the rete drawn from a place above the point *x* in Fig. 15. In this section many of the rete cells have entirely disappeared, which, with the widening of their interspaces, forms a small cavity. *bb*, rete cells in a condition of hyaline degeneration.

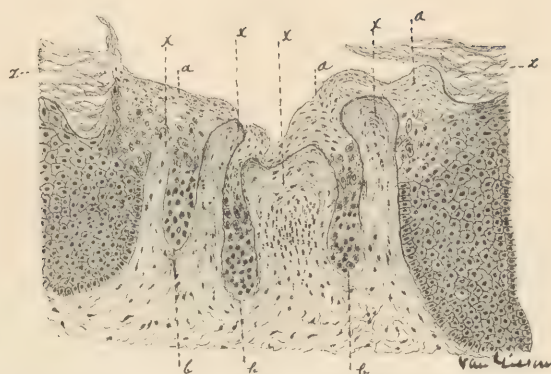


Fig. 18.—Showing the atrophic changes which may occur after an epidermis vesicle is completely formed. In this vesicle the upper wall has been removed or has desquamated, the contents have escaped, and the tissues surrounding the vesicle have a desiccated appearance. *aa*, dried-up or atrophic rete Malpighii, somewhat keratinized. *bb*, shrunken and degenerated interpapillary portions of the rete. *xxx*, dense and shrunken papillae. *zzz*, horny layer.



Fig. 17.—Illustrating a final stage in the production of the formation of the epidermis vesicles. The vesicle, filled with granular detritus and fluid, occupies the upper and middle layers of the rete Malpighii; the roof of the vesicle is bulged slightly upward; the rete cells beneath the cavity are degenerated and oedematous; the underlying papillae are infiltrated with small round and polygonal cells, which form two circumscribed clusters in the subjacent derma. This region of this vesicle is raised above the skin, and this vesicle formed one of the visible prurigo papules.



Fig. 19.—Same region as Fig. 18, showing the partial attachment of the upper wall of the dried-up cyst and the atrophic rete and papillae beneath. At *x* the cell clusters in the derma are very extensive.



Fig. 20.—Section showing the swollen endothelial cells of the sheath of Henle in a small nerve in the periphery of a group of swollen or proliferated connective-tissue cells in the lower portion of the derma.

FIG. 21.

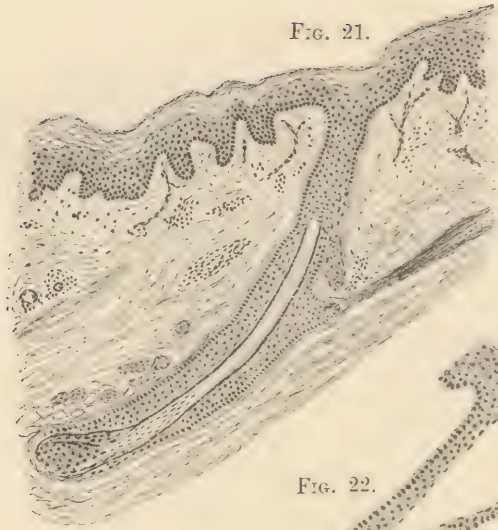
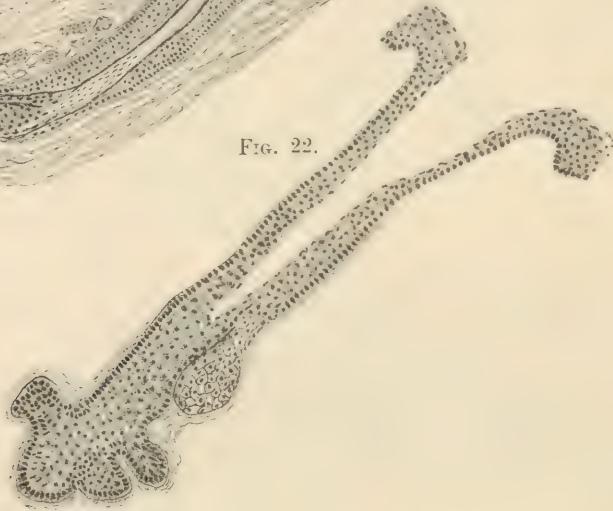


FIG. 22.



Figs. 21 and 22.—Sections of hair-follicles showing the pouch-like outgrowths of the outer root-sheaths, and the atrophied sebaceous glands. Fig. 21 shows the pigmented and beginning atrophic change in the papilla of the hair, and an outgrowth of the outer root sheath, just at the attachment of the hypertrophied arrector pili muscle.



FIG. 23.—A hair follicle showing the same changes as in Figs. 22 and 23, somewhat more highly magnified.

case, and was observed by me in one of my earlier cases. If I read authors aright, many of them are under the impression that the lesions of prurigo are of permanent character, and that they come to stay and to induce in the skin the well-known morbid changes. In the severest form of prurigo, of which I saw many years ago an interesting case in a man forty years of age, the tendency of the disease is certainly to a slowly continuous intensification of its lesions and their sequelæ. In this period of exacerbation, of which Fig. 3 presents a partial picture, the disease extended itself over the thorax and abdomen in the shape of a profuse eruption. With the increased number and distribution of the papules the child's sufferings were much intensified, and the evidences of scratching were plainly visible all over the trunk. Over the shoulders the picture strongly suggested pediculosis, while on the lower abdomen, buttocks, and thighs, the semblance to scabies in its papular form was very striking.

It was noted during the course of the disease that little scattered atrophic spots of a white color were quite often seen. These little lesions resulted from the involution of the prurigo papule, and it was evident from their examination that there had been a slight loss of tissue. As time went on, all traces of them were lost; so it is fair to suppose that no permanent deformity of the skin was induced. These spots may be seen represented on the back of the hand in Fig. 3, also along the forearm. I am not aware that attention has been called to them, since most of the existing descriptions of prurigo have been devoted to extreme phases of the disease. In his section of this essay Dr. Van Gieson describes the pathological appearances presented by these lesions.

I observed at various times a number of complications the nature of which is readily understood. Not infrequently wheals, large and small, waxy, white, and red, were produced by the child's vigorous scratching. So far as I could observe, these were only accidental, and were not the essential pathological forerunners of a papular outburst.

Then, again, evidences of bacterial infection were seen in scattered pustules, large and small, and ecchymatous crusts, which formed on the surface.

Besides these features, patches of eczema of ephemeral duration not infrequently showed themselves.

It is evident from the foregoing facts that during the eight months in which this case has been under my observation its characters and clinical features have undergone many changes and modifications, and I am certain that prior to the child's coming to me the same conditions existed. I frequently convinced myself that at certain times a diagnosis was very difficult and sometimes even impossible. I observed this same fact in the cases of the other two children suffering from prurigo seen by me.

The foregoing case is, as I have said, an excellent basis for study. In the three other cases seen by me and in cases belonging to others, I have seen more advanced and the most advanced characteristics of the disease. Prurigo supplies the essential groundwork for almost all forms of chronic inflammation of the skin. Therefore with the features of my case in mind it is easy to comprehend the more complicated cases. Progressing with various complications of long and short duration, the derma and epidermis in prurigo become thicker and denser. Then we see that pig-

mentation gradually sets in, that the surface lines become deeper and broader from the thickening of the horny layer, and that desquamation may be present in greater or less degree. When prurigo cases are complicated with chronic urticaria, a little time and careful watching will soon put the diagnosis beyond question. Then in the event of an eczematous complication, even if it covers the whole prurigo eruption, time and treatment will sooner or later make the diagnosis correct. I have several times been impressed with the necessity of prolonged observation in cases of chronic papular eczema with pale and even slightly red papules, which have been regarded by others as instances of prurigo. The coincident eruption can almost always be effectually cured; and then, if the case is watched, the features of prurigo (if that is the disease present) will soon again show themselves. In any case where an eczematous picture is presented and prurigo is suspected, a careful retrospect of the patient's history as far back as early years (if he or she or the guardians are ordinarily intelligent) will, I think, generally furnish facts which will enable the observer to determine whether the case began as prurigo or as eczema.

From this patient I excised two portions of skin during the time of a classical eruption of prurigo uninfluenced by any treatment, without any complication whatever, except that, perhaps, in some parts there were a few blood-crusts upon excoriated papules, and these were carefully avoided. The portions of skin contained typical prurigo papules in a presumably advancing and complete state. It will therefore be very distinctly understood that we have not submitted to study and examination any urticarial nodule or any concomitant or complicating lesion of the skin due to irritation or other accident. In other words, avoiding the errors which some authors have fallen into, clinically and microscopically, we have confined ourselves in this issue to investigating and portraying the morbid changes which take place in the skin in prurigo pure and simple.

MICROSCOPICAL EXAMINATION AND PATHOLOGY.*

The writers on the pathology of prurigo describe the changes in the skin quite uniformly, but their conclusions disagree so much and leave the subject so confused that I have studied the changes in this case independently of the work done by others, and shall describe, first, certain general changes in the skin not connected with the papules; secondly, the process of development of the papules which can be seen; and, finally, the results of the examination of the nodules underneath the epidermis which can not be seen with the naked eye but are perceptible to the touch.

The portion of the skin examined for the first two sets of changes was excised from the radial extensor surface of the left forearm. It measured about a half by three quarters of a centimetre in diameter, was stretched on cork and hardened in Müller's fluid and subsequently in alcohol. Serial sections cut by the celloidin imbedding process and stained double with hæmatoxylin and eosin were mounted partly in glycerin and partly in balsam. This piece of skin

* By Dr. Van Gieson.

contained two of the older prurigo nodules visible to the naked eye as very minute raised brownish points about a millimetre in diameter. One of these proved to be a crust or pellicle in the thickness of the horny layer pierced by a hair and lying over the mouths of three sweat ducts. The other was a cyst in the rete Malpighii.

I. THE GENERAL CHANGES IN THE SKIN NOT CONNECTED WITH THE PAPULES.

The epidermis is but little changed. The layers of the epidermis, except in a few places are regularly arranged and the topographical relations of the rete Malpighii are normal. Here and there the layers of the epidermis have an undulating outline, and the outer surface, instead of being smooth and even, is thrown up into alternating prominences and depressions. The only change in the epidermis is an irregular thickening of its layers. The rete Malpighii has a few places of localized thickening, such as shown in Fig. 4, but these are not at all extensive and are very few in number. The stratum corneum is slightly thickened in an irregular way. In some places it is normal and in other places, such as Fig. 5, there are circumscribed patches of the horny layer where the thickening is considerable in amount. A thickening of the stratum granulosum and an increase of the kerato-hyaline substance was not observed in the places corresponding to the thickened horny layer. The thickening of the horny layer seems to be due rather to an accumulation of the horny material by its not being removed from the skin rather than to an increased production.

The changes in the derma consist of scattered larger and smaller groups of small round and small polygonal cells encompassing the blood-vessels in the lower corium and at the lower margin of the pars papillaris (Figs. 4, 5, 6, 7, 15, 20, 23). Most of these cells in these groups appear to be derived from the connective-tissue cells of the derma lying close to the blood-vessels (Figs. 15 and 20). The papillæ are as a rule but little changed, but some of them are infiltrated to a moderate extent by the same small round and polygonal cells (Fig. 7). Some of these cell groups are clustered about the hair follicles as in Fig. 23, but the cell groups have no definite position to any of the annexa of the skin, but are scattered about all through the lower corium of the whole of the excised portion of the skin.

The sweat glands are normal except that several of them have slightly dilated mouths.

The hair follicles are very uniformly changed throughout the whole portion of the skin. There are one or more pouch-like outgrowths of the outer root sheath generally situated at the lower portion of the follicle. The outer root sheath is quite uniformly pouched out at the attachment of the arrector pili muscle, which is hypertrophied. A few of the follicles with their shafts are shrunken, indistinct, and pigmented, and some of the follicles show evidences of the formation of lanugo hairs (Figs. 7, 21, 22, and 23).

The sebaceous glands are all considerably smaller than normal, although their constituent elements are but very little changed.

The small nerve trunks in the skin were carefully examined in all of the sections with the oil-immersion lens and compared with those in the normal skin, and were found unchanged, except in one or two places, when situated in or near the cell clusters in the derma. In one or two of the small bundles thus situated there is a limited swelling or proliferation of the endothelial cells of the sheath of Henle (Fig. 20).

The endothelial cells in the *lymph spaces* or small veins of the pars papillaris at or near the cell clusters in a few places are swollen and granular (Fig. 15).

Some of the prurigo papules are due to small cysts in the epidermis, and others apparently correspond to small crusts or scab-like pellicles in the thickness of the horny layer.

II. DESCRIPTION OF STAGES OF DEVELOPMENT OF PRURIGO PAPULES DUE TO EPIDERMAL CYSTS.

The earliest stage of the development of the epidermic cysts or vesicles is the widening of the intercellular spaces in several of the lowermost cells of the rete Malpighii covering an œdematous papilla. This widening of the cell spaces appears to be due to the exudation of fluid from the subjacent papilla (Figs. 8, 9, and 10). Besides the distention of the lowermost rete-cell spaces by fluid, there is a liquefactive degeneration of the protoplasm of the cells. The portion of the cell body about the nucleus apparently becomes converted into fluid, and appears as a light halo about the nucleus, while the periphery of the cell stains deeply (Figs. 8, 9, 10, 11, and 12). In a more advanced stage of this degeneration of the rete cells the liquefaction of the central protoplasm increases so that the nucleus lies in a cavity surrounded by a mere shell of the former cell body. Finally, the nucleus and remains of the cell body disappear or become converted into granular material (Figs. 13, 14, 15, and 16).

A portion of the cell bodies is also converted into a hyaline or homogeneous shining substance easily recognized by its intense affinity for eosine (Figs. 11, 12, and 13).

By reason of the combination of these two processes—the distention of the rete-cell spaces and the liquefactive degeneration of the cells themselves—due to the soaking of fluid into the rete from the subjacent œdematous papillæ, minute branching or irregular-shaped cavities are produced, and Figs. 8 to 17 show so well the stages of development from the smaller to the larger cavities that a more detailed description of their formation can be dispensed with.

Each of these little groups of degenerated rete cells or cavities can always be traced to an œdematous papilla just beneath the cavity, or in the neighborhood of the cavity. Figs. 11, 12, and 15 show the relations of the œdematous papillæ to the degenerated groups of rete cells or cavities.

The degenerated or cystic spots in the rete Malpighii produced by the œdematous papillæ do not occur at all frequently in the sections. It is only here and there, in many sections, that the incipient cavities and corresponding œdematous papillæ can be found. The places from which the drawings Figs. 8 to 16 were taken were

scattered about in fifty different sections without having any particular definite relation to the other changes in the skin. In one place, however (Fig. 15), the base of the œdematous papilla is infiltrated with apparently proliferating connective-tissue cells surrounding a small vein. The position of this small vein at the base of the papilla is somewhat suggestive as to the source of the fluid. It is possible that the fluid exuded from the smaller papillary branches of this vein or from the vein itself.

In other places the derma beneath the œdematous papillæ, as in Figs. 11 and 12, is quite normal and not infiltrated with the small round and polygonal cells.

After having detailed the intermediate stages in the development of the epidermis cysts, a final stage of their development, such as is shown in Fig. 17, may be described. Such a vesicle as this becomes visible to the eye as a minute raised, brownish point, as shown in the photographs of the forearm. In this advanced stage of the prurigo papule produced by a large epidermis cyst, the superior wall of the cavity is formed by the slightly condensed or thickened stratum lucidum and granulosum, and the lateral and inferior walls are formed by the rete mucosum the cells of which below the cavity are indistinct and do not form the usually sharp boundary line with the pars papillaris. The rete cells below the cavity are degenerated as previously described, and have very much widened interspaces (Fig. 17). The cavity is situated in the middle and upper layers of the rete Malpighii and contains one or two cells looking like leucocytes, hyaline droplets, fragmentary nuclei, and the detritus of destroyed rete cells. The pars papillaris beneath the cavity contains more extensive clusters of small round and polygonal cells than elsewhere in the section.

This vesicle, then, is a later member of the series of changes shown in Figs. 8 to 16, and has become large enough to produce one of the visible prurigo papules.

The Retrogressive Changes which may occur after the Epidermis Vesicle is fully formed.—These changes I am unable to describe completely, because in the limited supply of material only one papule was found in this condition.

This retrogressive or atrophic change in the vesicles is a kind of desiccation of the rete Malpighii surrounding the cavity, and an atrophy and condensation of the underlying papillæ, occurring after the superior wall of the cavity is picked off or desquamates or the fluid of the cavity otherwise escapes to the surface. Fig. 19 shows a cyst like the one in Fig. 17, but the superior wall of the cavity is partially removed, permitting the escape of the contents of the vesicle. In Fig. 18 the superior wall is entirely absent and the vesicle has become dried up, leaving a minute atrophic spot in both the epidermis and papillæ. The rete cells about and beneath the exposed cavity are desiccated, degenerated, and partially keratinized. The interpapillary portions of the rete are shrunken and indistinct and the papillæ are dense and shrunken. Beneath the atrophied papillæ there is a very extensive cluster of small round and polygonal cells (Fig. 19).

In this way a small atrophic portion of the epidermis with its underlying papillæ may be produced corresponding to the site of the former epidermis vesicle. Such atrophic

spots as these ought to show in the skin in the living subject of prurigo, and I think they correspond to the tiny white spots in the photographs.

Description of Certain of the Prurigo Papules corresponding apparently to Small Crusts or Scab-like Pellicles in the Thickness of the Horny Layer.—These are minute pellicles of granular material containing fragmentary nuclei surrounded by a thin envelope of concentrically arranged cells somewhat like those of the stratum lucidum and are situated in the midst of the horny layer (Figs. 6 and 7). The scab shown in the figures is pierced by a hair and lies over the orifice of at least three sweat ducts. This crust looks in several sections as if it pressed down on the rete (Fig. 3), because the interpapillary portions of the rete are thrust aside laterally.

The rete is perfectly intact beneath the scab, and I could not determine the origin of this scab, for only one of them was found, and this one gave no clew to its source unless it was due to an altered deposit of the secretion of the atrophied sebaceous gland of the hair passing through the crust. This does not seem probable, however, for there are many damaged sebaceous glands in the sections without these crusts or scab-like pellicles.

III. THE EXAMINATION OF THE HARD, INVISIBLE SUB-EPIDERMAL NODULES PERCEPTIBLE TO THE TOUCH ONLY.

For this purpose a piece of skin, similar in size and position to the first piece, containing two of these nodules, was carefully hardened in osmic acid and the positions of the nodules carefully noted, but, in examining all of the sections through the entire region of the nodules, I could find no trace of them whatsoever. The two nodules disappeared in the hardening. This piece of the skin was almost normal with the exception of the cell clusters in the derma, which were less extensive than in the first piece. The hairs, arrector pili muscles, and sebaceous glands were changed as already described.

RÉSUMÉ AND CONCLUSIONS.

With the exception of the systematic and conscientious paper by Morison (*Amer. Jour. of the Med. Sci.*, 1883, p. 341) and a good but somewhat diffuse article by the most recent writer on the subject, Kromayer (*Archiv f. Derm. u. Syph.*, 1890, p. 77), the deductions in the pathological literature of prurigo are largely speculative and contradictory, and at any rate fail to give a clear, concise, definite theory of the formation and course of the prurigo nodules and papules in harmony with the anatomical facts.

Knowing very well how much it hinders the progress of knowledge of a disease to make conclusions and hypotheses unconfirmed by experimental research or not warranted by the number of accurately described anatomical facts discovered about the disease—and this is decidedly the case with prurigo—I think it more appropriate for me to state the anatomical facts in this case carefully than to make any general deductions except of the most guarded and limited kind.

The anatomical processes and changes in the skin in this case seem to be as follows. There is a chronic inflammation of the derma, most extensive just below the pars

papillaris, although the latter, with its papillæ, is also affected in places. This inflammation is mainly of the chronic cellular variety, produced by a proliferation of the connective-tissue cells just about the blood-vessels, although it is not unlikely that an exudation of leucocytes from the blood-vessels may play a minor rôle in the production of some of the dermic cell clusters. Along with this chronic cellular inflammation the hair follicles, sebaceous glands, and smooth muscle fasciculi become changed secondarily, or by sharing in it, as in some other chronic inflammations of the skin—as, for example, in lichen ruber—and are not in any way the primary or essential lesions of the disease. The alterations in the small nerve bundles, as shown in Fig. 20, are simply due to the fact that this portion of the nerve happened to run through a patch of the proliferating connective-tissue cells, and the endothelial cells in Henle's sheath are swelling and proliferating just as the surrounding dermic connective-tissue cells.

From the fact that the traces of the subepidermal nodules disappeared so completely in the hardening, it seems reasonable to suppose that they were occasioned by a circumscribed portion of the derma, distended by fluid, which was abstracted by the hardening fluids, and that the shrinkage of the hardening fluids obliterated the spaces in the derma where this exudation of fluid had been during life. This seems the more plausible from the fact that some of the papillæ do show plainly enough evidences of distention by fluid, as already mentioned.

As Kromayer suggests, it would be well to examine these subepidermal nodules as well as the other prurigo papules in the fresh condition. If the subepidermal nodules are produced by a circumscribed effusion of fluid in the cutis, frozen sections might throw considerable light on this point.

Although the material has been limited, and from one case only, I think that the prurigo nodules and papules are formed in the following way: A circumscribed effusion of fluid in the deeper derma, or possibly involving a group of fifteen or twenty papillæ, produces subepidermal nodules. In the course of time this fluid gradually makes its way from the papillæ into the overlying rete Malpighii and filters through the intercellular spaces. This fluid can not escape through the outer surface of the epidermis on account of the density of the stratum lucidum; thus being held in the rete Malpighii, the effusion gradually destroys the rete cells and produces a cyst which finally becomes large enough to raise up the stratum lucidum so that it is visible to the naked eye as one of the prurigo papules. In this way the subepidermal nodules which appear first make their way to the surface of the skin and produce the later visible papules.

When the upper wall of the cyst peels off or is removed, or the contents of the cyst otherwise escape, the damaged and soaked rete and papillæ beneath become changed so that a minute atrophic spot is left in the skin.

What relations the subepidermal nodules have to the cysts, or whether the cysts develop later from the nodules, or if the cysts form independently of the nodules, I could

not determine from the sections alone, but it seems likely that the cysts develop from the nodules, as observed clinically.

Prurigo nodules and papules, then, seem to be due to a circumscribed effusion in the deeper derma or papillæ which soaks into the rete and produces cysts, which may subsequently atrophy, and is accompanied by a chronic cellular inflammation in the derma with secondary alterations in the hairs, sebaceous glands, smooth muscle bundles, and hypertrophic regions in the epidermis.

This explanation, however, of the formation and course of the prurigo nodules and papules leaves open the question of the relationship of the scattered circumscribed exudations of fluid to the chronic cellular inflammatory process. There seems to be no way of determining from the sections whether the exudation of fluid from the vessels occurs from the vessels first, and, by supplying increased nutrition to the perivascular connective tissue, causes them to proliferate, or whether the exudation of fluid is secondary to, or part and parcel of, the chronic perivascular cellular inflammation. Morison found in the subepidermal nodules a focus of small round and polygonal cells rather more extensive than elsewhere.

It seems to me that it is much wiser to defer the solution of the question of why it is that this exudation occurs until we find out more about the causes of inflammation in general, than to speculate upon it with the confusing terms idioneurosis, vaso-motor troubles, etc., as some of the writers have done.

The element of the results of scratching was eliminated in the portions of skin excised by Dr. Taylor.

The changes in the skin have some analogy to those in lichen ruber, and the characteristic features of chronic cellular inflammation in general—viz., its slow course, its tendency to persist for a long time, and its proclivity toward exacerbations—agree well with the clinical history of the disease.

RÉSUMÉ OF PATHOLOGICAL LITERATURE.

Up to 1883 this literature is so clearly discussed by Morison that any allusion to it here is superfluous. Since Morison's paper the subject has been dealt with by Riehl (*Arch. f. Derm. u. Syph.*, 1884, p. 41), Leloir and Tavernier (*Annales de dermat. et de syph.*, 1889, No. 7), and Kromayer (*loc. cit.*). Riehl has been criticised for having mistaken his case for one of urticaria, and at any rate his description of the papules indicates that it was not a typical case of prurigo. Leloir and Tavernier's work is of the careless, gossiping kind, and does not indicate that enough time and careful study had been spent on the work to make their conclusions at all acceptable. They describe older vesicles without stating how they are formed, and indicate a preference for the view that the vesicles are produced through the agency of the sweat ducts. Kromayer states that hardened material is not suitable for examining the changes in prurigo—and I agree with him in this, especially in the subepidermal nodules—and thinks that an exudation of fluid in the upper layers of the cutis is the primary lesion in prurigo.

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